The Journal of HEREDITY

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Nikolai Koltzoff — An Appreciation

More Medico-Genetic Terms

Sex and Utopia

The Multiple-Spurred Fowl

Color Patterns in Grasshoppers

Taste Reactions of the Dionne Quintuplets

VOLUME 32 OCTOBER, 1941 NUMBER 10

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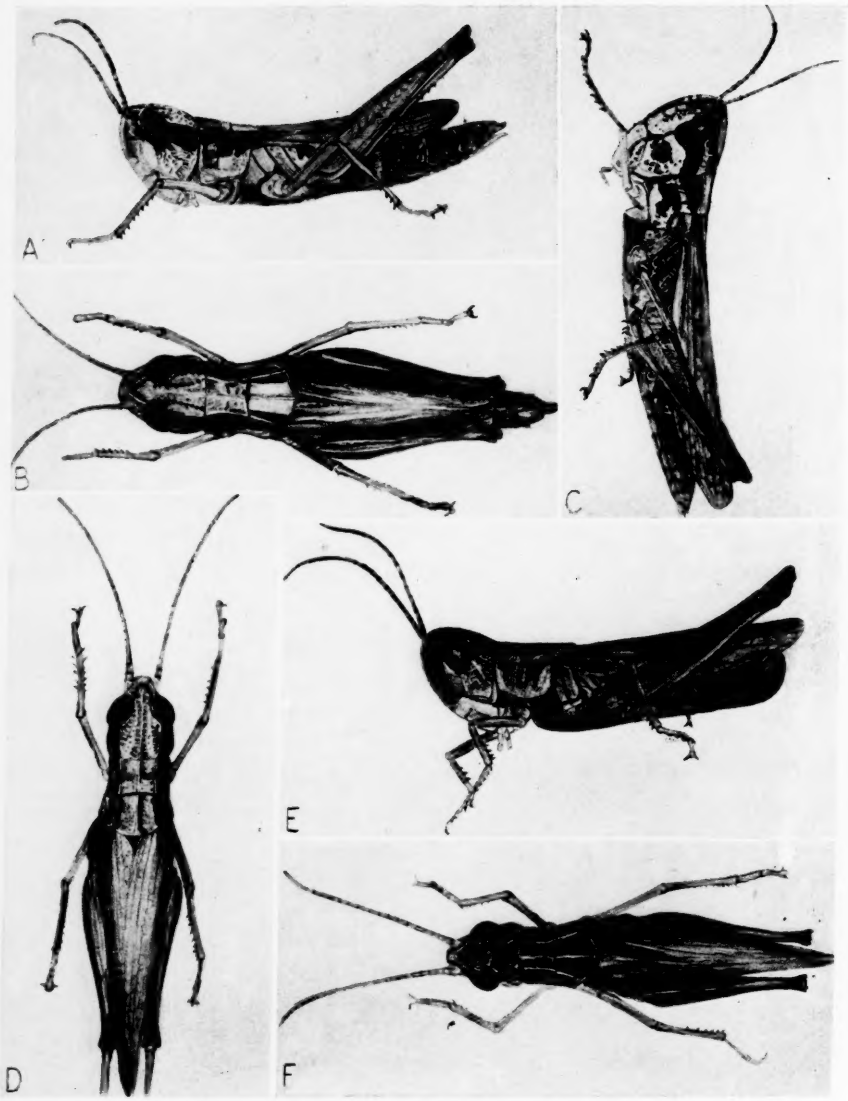

COLOR PATTERNS IN CHORTHIPPUS LONGICORNIS

Frontispiece

A, B—"Plus" type, s^{+}. *C*—Differs from "plus" type in striped pattern on side of gena and pronotum, s^{V}. *D*—Differs from "plus" in having green sides with narrow black dorsal stripe, back same as in "plus", s^{L}. *E, F*—Differs from "plus" in having green sides with narrow black dorsal stripe, as in s^{L}, and brownish red back, s^{R}.

GENETIC STUDIES ON CHORTHIPPUS LONGICORNIS

MARGARET CREIGHTON AND W. R. B. ROBERTSON*
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THE genetics of the grouse locusts has been studied extensively by Nabours et al.[9] Otherwise little work has been done on inheritance in Orthoptera. Fryer[5] studied the inheritance of two characters in *Clitumnus cuniculus*, a bisexual Phasmid. Hancock[6] and Nabours[10] found that pink and green coloration were inherited as a pair of mendelian characters in the katy-did, *Amblycomorpha oblongifolia*. King[8] found that melanism was inherited as a recessive autosomal character in *Melanoplus differentialis*. Carothers[3] has studied the inheritance of several characters in a species cross between *Trimerotropis citrina* and *T. maritima*. Sansome and LaCour,[11] working with *Chorthippus parallelus*, described the action of fourteen pairs of genes. The present study concerns the inheritance of four color patterns in *Chorthippus longicornis*, which has been identified with *Chorthippus parallelus* by Hebard.[7]

Life Cycle

The wild grasshoppers used in this study were collected in several widely separated localities in Iowa; Iowa City, Lake Okoboji, and Devil's Backbone State Park. In the laboratory they were kept in wire cages with wooden frames and fed on grass and lettuce in the summer and on wheat seedlings, lettuce, and bran mixed with a little cod-liver oil in winter. The females deposited their eggs in a mixture of sand and soil contained in glass tumblers. The eggs are laid in pods which may contain from one to eight eggs, and one female may lay twelve or more pods. The eggs are undergoing maturation at the time of laying. Development begins immediately and continues for approximately three weeks. At this time the embryos are in a developmental stage similar to that described for *Melanoplus differentialis* by Slifer.[12] At the end of the three-week period development stops and the embryos enter diapause, which may continue indefinitely if the eggs are kept at room temperature. It can be broken, however, by subjecting the eggs to low temperature for a month, as shown by Bodine.[1,2] When the eggs are returned to room temperature, development resumes and hatching occurs in from ten to twenty days.

The young hoppers moult on hatching and four times thereafter. They reach maturity at about one month after hatching. The time of development varies considerably with temperature. The intervals given are the average for room temperature.

The mortality among young animals in the laboratory was rather high. This combined with low fertility of laboratory-bred animals made it difficult to maintain lines successfully and to obtain homozygous stock.

Parthenogenesis is a common phenomenon. Isolated virgin females will nearly always deposit eggs. The embryos develop with varying degrees of abnormality and usually die before hatching. A few have hatched, however, and one lived through the second instar. It has been shown by Creighton[4] that the unfertilized eggs undergo both maturation divisions and that doubling of the chromosome numbers occurs somehow in the cells of the developing embryo.

Sex could be determined at hatching by the presence of the primordia of the ventral valves of the ovipositor in the females.

The length of the wings is highly vari-

*The manuscript was practically completed at the time of Dr. Robertson's death on March 15, 1941.

able. Animals collected in the wild usually have short elytra while among those raised in the laboratory there is a much higher frequency of long elytra. The two environments differ in many respects but it seems likely that the higher temperature of the laboratory is responsible for the longer wings.

Inheritance of Color Patterns

The color pattern most commonly found in the wild has been designated the "plus" type. The general color of the dorsal and lateral surfaces of the head and pronotum in this type varies from light greenish gray to yellowish gray. The black pigment is distributed on the side of the pronotum and head as shown in Frontispiece *A*. The vertex is mottled in black and gray and there is a wedge-shaped band of black on each side of the dorsal metazona (Frontispiece *B*). The extent and intensity of the black pigment is highly variable and the variability seems to be at least partly under the control of the environment.

The relationship of three other color patterns to the "plus" type and to each other has been studied. The patterns are symbolized as follows:

s^+ — "plus" type.

s^V—Variegated side (Frontispiece *C*). The black pigment on the side of the pronotum is distributed in a more or less striped pattern and there is a patch of dense black mottling at the anterior ventral corner of the gena. The pattern on the dorsal surface is variable, depending apparently upon other genetic characters, as well as upon environmental conditions.

s^L—Light back, green side (Frontispiece *D*). The gena and side of the pronotum are bright to dull green with a narrow black dorsal border as in Frontispiece 5. The vertex and dorsal surface of the pronotum are light gray or yellowish gray.

s^R—Red back, green side (Frontispiece *E*, *F*). The gena and side of the pronotum are the same as for s^L but the vertex and dorsal surface of the pronotum are brownish red. This color may vary from light reddish tan to dark red, to dark brownish red, to almost black.

Various types of matings were made between animals showing these patterns. The results are given in Table I. They indicate that the four patterns considered form a series of multiple alleles, and that the order of dominance is as follows: s^R, s^L, s^V, s^+.

It is possible that the s^R pattern is determined by two closely linked genes and that the s^L pattern is the result of a crossover between s^R and the "plus" type. This possibility is supported by the fact that animals with reddish backs and more or less "plus" type sides are found in nature. The genetics of this character has not been worked out, however, because of extreme variability in the expression of the red color, and its relation to s^R is, therefore, not known. If the s^R and s^L patterns are determined by two genes the linkage must be very close, since in 551 gametes tested, there have been no crossovers.

Discussion

Since *Chorthippus longicornis* has been identified with *C. parallelus* by Hebard,[7] it is interesting to compare the observations here presented with those of Sansome and LaCour[11] on *C. parallelus*. There are several outstanding differences.

1. The maximum number of eggs in an egg pod in the Iowa form is eight. Sansome and LaCour found about sixteen eggs per pod.

2. The time from hatching to the final moult is about one month in the Iowa form with the average instar duration about one week. The form used by Sansome and LaCour has a ten week growth period with an average instar duration of fourteen days. This difference may in part be due to differences in temperature.

3. Long elytra appear to be associat-

Table I. Distribution of offspring of various types of matings. Number expected on mendelian segregation in parentheses.

Mating	Phenotype of offspring			
	s^R	s^L	s^V	s^+
s^R/s^R x s^+/s^+	19			
s^R/s^R x s^R/s^+	301			
s^R/s^+ x s^R/s^+	266 (265.5)			88 (88.5)
s^R/s^+ x s^+/s^+	189 (187)			185 (187)
s^L/s^+ x s^L/s^+		60 (60)		18 (20)
s^L/s^+ x s^+/s^+		10 (11.5)		13 (11.5)
s^V/s^+ x s^V/s^+			52 (57)	24 (19)
s^V/s^+ x s^+/s^+			163 (156)	149 (166)
s^R/s^+ x s^L/s^+	132 (130.5)	67 (66.25)		65 (66.25)
s^R/s^L x s^L/s^+	20 (21)	22 (21)		
s^R/s^L x s^+/s^+	39 (35.5)	32 (35.5)		
s^R/s^+ x s^V/s^+	96 (71)		30 (45.5)	46 (45.5)
s^R/s^R x $s^V/?$	19			
s^R/s^V x s^+/s^+	67 (65.5)		49 (45.5)	
s^L/s^+ x s^V/s^+		17 (15.5)	5 (7.75)	9 (7.75)

ed with high temperatures and a consequent short developmental period in the Iowa form. Sansome and LaCour found that long wings were associated with a long developmental period and abnormality of the gonads.

Concerning the relationship between the color patterns of the Iowa and British forms, little can be said with the information available. It is possible that the "conventional type" of Sansome and LaCour, which is green on all dorsal surfaces, is identical with s^L and s^R with regard to the pattern on the gena and side of the pronotum. Other genes make the dorsal surface green. The *"gb"* of Sansome and LaCour, which makes the dorsal surface of the pronotum gray, may, therefore, correspond to s^L. If this is true, it becomes more likely that s^R and s^L are both the result of two closely linked genes acting independently on the lateral and dorsal surfaces.

Summary

1. The inheritance of certain color patterns of *Chorthippus longicornis* was studied. Four patterns were found to be determined by a series of multiple alleles.

2. Certain features of the breeding habits, life cycle, and color patterns are compared with those described by Sansome and LaCour for the British form of the same species. The British form shows marked dissimilarity in certain respects to the form found in Iowa.

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11. Sansome, F. W., and L. LaCour. The genetics of grasshoppers: *Chorthippus parallelus*. *J. Genet.* 30:415-422. 1935.

12. Slifer, E. H. Insect development. IV. External morphology of grasshopper embryos of known age and temperature history. *J. Morph.* 53:1-9. 1932.

Literature of Waddington's Chick Twin Article

THROUGH a blunder in making up the August issue, the literature citations were omitted from Dr. C. H. Waddington's article "Twinning in Chick Embryos," pages 268 to 270 of the August 1941 issue. These are as follows:

Literature Cited

1. Abercrombie, M. The Behaviour of Epiblast Grafts beneath the Primitive Streak of the Chick. *Jour. Exp. Biol.* 14:302. 1937.

2. ———— (Unpublished).

3. Abercrombie, M., and C. H. Waddington. The Behaviour of Grafts of the Primitive streak beneath the Primitive Streak of the Chick. *Jour. Exp. Biol.* 14:319. 1937.

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5. Twiesselman, F. Experiences de Scission Précose de l'Aire Embryogège de Scis-Poulet. *Arch. Biol.* 49:285. 1938.

6. Waddington, C. H. Induction by the Endoderm in Birds. *Arch. Entw.-mech.* 128: 502. 1933.

7. Waddington, C. H., and A. Cohen. Experiments on the Development of the Head of the Chick Embryo. *Jour. Exp. Biol.* 13:219. 1936.

8. Waddington, C. H., and G. A. Schmidt. Induction by Heteroplastic Grafts of the Primitive Streak in Birds. *Arch. Entw.-mech.* 128: 522. 1933.

A reprint containing the citations will be furnished on request. The editor regrets this unfortunate error.

"KNOTTED" MAIZE PLANTS

Figure 1

"Knotted" maize plants showing the extreme and the slight manifestations of this condition. "Knotted" is dependent on a single dominant gene located in the first linkage group.

HERITABLE CHARACTERS IN MAIZE

51—"Knotted Leaf"*

A. A. Bryan† and J. E. Sass‡

NORMAL AND KNOTTED LEAF PLANTS

Figure 2

The first plant on the left and the sixth from the left are normal. It is probable that the degree of knotting, of which three stages are recognized, may be due to modifying factors. The extremely deformed plants are generally homozygous for the *Kn* gene.

IN an ear-row planting of the open-pollinated variety Walden Dent made in 1922, a plant character was observed that has been called "knotted leaf" and given the symbol *Kn kn*. The row in which this character was noted was one of 500 such ear rows planted at the inauguration of the present corn breeding program at Ames, Iowa. No similar character has appeared in other stocks included in the breeding program. Tests indicate this character to be a dominant.

Knotted leaved plants are distinguish-

*Contribution from the Farm Crops Subsection and the Botany and Plant Pathology Section, Iowa Agricultural Experiment Station, Ames, Iowa, and the Division of Cereal Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture, cooperating. Journal Paper J-855, Project 182.

†Former Agronomist, Division of Cereal Crops and Diseases, and collaborator Agronomy Section, Farm Crops Subsection, Iowa Agricultural Experiment Station. Grateful acknowledgment is made to Dr. G. F. Sprague, who prepared the genetical portions of the manuscript after the death of the senior author.

‡Assistant Professor, Botany Department, Iowa State College.

able from their normal sibs when the plants reach a height of two or three feet. In general knotted plants are somewhat shorter, and later than their normal sibs and exhibit characteristic "knots" on the leaf blades and sheaths (Figures 1 and 2). The character is somewhat variable in its expression. Some plants have only a few scattered knots on the leaves. In others the upper leaves may be so severely affected that the tassels emerge very late or may even fail to emerge. Slightly and extremely knotted plants are illustrated in Figure 1.

"Knotted leaf" is inherited as a simple dominant. The progeny of eight selfed plants classified as slightly knotted consisted of 293 *Kn* and 88 *kn* plants. Crosses between heterozygous knotted and normal plants produced 193 *Kn* to 229 *kn* plants. Expectation on the basis of a backcross ratio was 211, the deviation being 18 ± 6.9. In general plants exhibiting an extreme type of knotting have proved to be *Kn Kn* in constitution when selfed or outcrossed. However, the degree of expression of this character cannot be attributed solely to the homozygous or heterozygous condition.

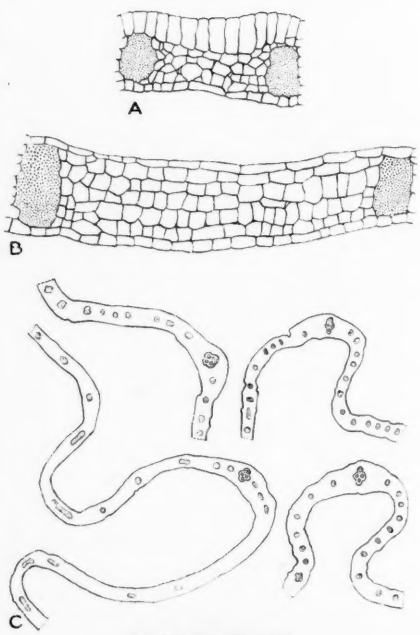
Knotted plants were arbitrarily grouped into three classes on the basis of the amount of leaf knotting and all possible intercrosses made among these types. Intercrosses among slightly knotted plants produced progeny that were preponderantly of this same class. Similarly intercrosses among extremely knotted plants produced progeny that were classed as intermediate or extreme for knotting. Such results suggest that modifying factors may determine a portion of the variability in expression of this character.

Linkage tests indicate that the knotted leaf character belongs in the first linkage group. The data obtained in 1937 are presented in Table I.

These data indicate that *Kn* is located between f_1 and bm_2. They also indicate that f_1 is to the left of *br* which is not the accepted linear order.

Somewhat more extensive data were obtained from a similar backcross in 1938. The data are presented in Table II.

The linear order with the recombination percentages is *P* 49.2 *br* 7.1 f_1 31.6 *Kn* 23.5 bm_2. The 1937 and 1938 data are not in agreement as to the order of *br* and f_1 but clearly indicate that *Kn* is between f_1 and bm_2 and somewhat closer to the latter.



LEAF SECTIONS

Figure 3

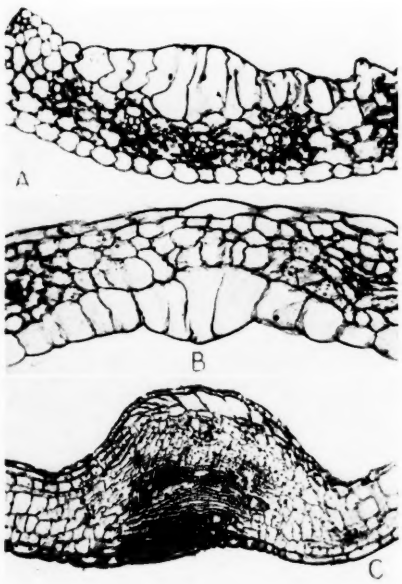
A—Interfascicular distance in a normal leaf compared with *B*, the expanded interfascicular portion of blade in a knot (× 184). *C*—Outlines of sections of leaf knots of various sizes (× 25). The dorsal (outer or lower) surface can be located by the position of the phloem, stippled in the larger veins.

Histological Studies

Material and Methods

Plants were grown in the greenhouse for these studies. Leaf knots were excised at intervals, beginning with knobs that were just visible to the naked eye, and sampling was continued until the leaves were expanded fully. The tissues were killed in the following formula:

1 per cent chromic acid	20 c.c.
1 per cent acetic acid	10 c.c.
37 per cent formaldehyde	10 c.c.



CELL STRUCTURE

Figure 4

A—Transverse section showing excessive enlargement of bulliform cells. *B*—Longitudinal aspect of enlarged cells of lower epidermis. *C*—Longitudinal section of small knot showing displaced tracheal elements of distorted vascular bundle (× 184).

The material was embedded in paraffin, and sectioned transversely and longitudinally. Semi-diagrammatic drawings were made by microprojection.

Observations

Examination of sections under low magnification shows that a "knot" is a fold in the leaf. As shown in Figure 3*C*, the convexity may occur on either the upper (inner, ventral) or on the lower (outer, dorsal) surface. There is very little increase in the thickness of the blade. Thus, most of the distortions, especially the large ones, are not emergences on the leaf, but are wrinkles and folds.

Distortions of the leaf are associated with distinctive histological changes. The number of layers of cells comprising the thickness of the leaf is usually identical in normal and knotted regions. A few localized regions of knotty tissue show evidences of periclinal cell divisions (parallel with the surface), whereby the number of layers of cells is increased. It is clear, however, that the folds or knots are not produced by thickening or periclinal cell proliferation. Extensive proliferation occurs by anticlinal cell division (at right angles to the surface). Epidermal cells and vascular elements do not undergo extensive division, but the inner cells of the mesophyll undergo repeated division. The cell walls are laid down in an accurately anticlinal plane, producing a regular stratification (Figure 3*B*). The result of this proliferation of the mesophyll is a marked lateral expansion of the interfascicular portion of the leaf blade in the knotty regions. Figure 3 affords a comparison of the structure and dimensions of typical interfascicular tissues in normal areas (*A*) and distorted areas (*B*).

Resistance to lateral expansion of proliferated regions is provided by the normal leaf blade surrounding the proliferating regions. Consequently, the proliferating portions of the blade are forced into wrinkles, folds and knobs, producing the characteristic "knots." The epidermal cells undergo considerable lateral expansion or stretching, and become somewhat narrowed parallel to the surface.

Distortion of epidermal cells in localized regions is associated with the formation of knots. The bulliform or motor cells undergo considerable enlargement, especially anticlinally. The cell walls probably become attenuated and very thin. These cells collapse laterally and become wedge-shaped (Figure 4*A*). Similar enlargement of groups of cells of the lower epidermis also takes place (Figure 4*B*). It is not improbable that this process contributes to the formation of folds in the leaf.

The vascular bundles in the knots appear to be normal in structure in most sections. Many longitudinal sections of knots show considerable lengths of vascular bundles, exhibiting unbroken and undistorted vascular elements throughout the bundle, but showing proliferated stratification of the mesophyll parenchy-

ma. This absence of xylem breakage indicates that proliferation occurs in the parenchyma and procambium cells of very young leaves, and that considerable expansion of the knots occurs before the tracheae become differentiated and firm.

A few of the small knots do not seem to be of the folded type, but are emergences on the ventral (upper) surface of the leaf (Figure 4*C*). Irregular proliferation of parenchyma is evident, and displaced tracheal segments occur. The absence of mechanical breakage in the mature tracheal elements indicates that the distortion occurred in the procambial strands of incipient knots.

The foregoing observations lead to the conclusion that these abnormalities are the result of localized proliferation of mesophyll parenchyma and very restricted proliferation of procambial cells in the meristematic region of the leaf; subsequent expansion of these regions, acting against the normal areas, produces the described distortions.

Table I. Data obtained in 1937 from the backcross

$$\frac{+\ +\ \frac{cn}{+}}{br\ f_1\ +\ \frac{+}{brg}} \times br\ f_1\ kn\ brg.$$

Parental Combinations		Recombinations in the Region Indicated																							
		1		2		3		1, 2		1, 3		2, 3		1, 2, 3											
165	182	14	4	50	59	47	52	7	10	1	2	16	32	6	2										
347		18		100		99		17		3		48		8											

Table II. Data obtained in 1938 from the backcross

$$\frac{+\ br\ f_1\ +\ \frac{bm}{+}}{p\ +\ +\ \frac{+}{kn}} \times p\ br\ f_1\ kn\ brg$$

Parental Combinations		Recombinations in the Region Indicated																							
		1		2		3		4		1, 2		1, 3		1, 4											
139	122	140	145	9	11	99	41	42	61	9	11	53	21	51	52										
281		291		20		140		95		20		144		103											

Recombinations - Continued																									
2, 3		2, 4		3, 4		1, 2, 3		1, 2, 4		1, 3, 4		2, 3, 4		1, 2, 3, 4											
7	1	8	2	32	15	9	4	0	4	4	23	0	3	2	2										
8		7		37		13		4		27		3		4											

✦

UTOPIA OR SOMETHING

THE late Mr. Unwin was one of those sanguine souls who undertake great projects in human understanding. Before the present volume* was begun he had completed an ambitious study of sex and society. He seems to the reviewer to find complicated things simple, . . . illusionarily simple. In this posthumous volume, which sets forth the basic blueprint for Unwin's brave new world, two things are put forward as having prevented man from reaching even the suburbs of Utopia. They are money and sex. Or put it another way, eating, sleeping and dressing, recreating and reproducing. That covers pretty much everything. Mr. Unwin ventures hopefully forth on this vast ocean of complexities and steers himself in less than five hundred pages into a permanent haven of rest.

The trouble with money is easily solved. There is a corporal's guard of major follies in ownership and symbolism. Land is not a commodity, and gold is not a basis of value—it is a symbol. Future roses around Fort Knox may bloom the yellower because a dead symbol lies buried there. The discussion is lengthy, but the Gordian Knot is as simple as learning how to use a check book in new and alluring ways. The reviewer is no economist, but if he understands it correctly everybody is to get a great big checkbook and plenty of ink. After the bank paper and writing fluid goes dry he gets some more. It is not quite as simple as that, but almost. If you want to build a ship, you ask for a check book and get one. Then you write checks for materials and labor and other essentials and get your boat right away. Then you use the boat busily and retire your checks with the income derived from it. As soon as everything is paid off a government inspector (or a hostile U-boat?) sinks the boat and you start all over again. If you cheat and keep on using the boat after its paid off, there's

(*Continued on page* 350)

*Hopousia, or The Social and Economic Foundation of a New Society, by the late J. D. Unwin, with an introduction by Aldous Huxley, and a preface by Y. J. Lubock. Pp. 475, $5.00, Oscar Piest, New York. 1940.

DR. NIKOLAI K. KOLTZOFF

1871-1940

ON December 2, 1940, Dr. Nikolai Konstantinovich Koltzoff died in Leningrad in his sixty-ninth year. At the time of his death he was professor at two universities and Director of the Institute of Experimental Biology. He was a former Director of the Central Station of Genetics.

Koltzoff was appointed a privatdozent in Moscow University and defended his dissertation in 1894. His first paper dealt with the skeleton of the fins of teleostean fishes and was published in Moscow. Later he was engaged in the study of metamersim in the head of Petromyzon. He published a short preliminary note on this subject in *Anatom. Anzeigher* in 1899. His 230-page work on the same subject published in Moscow in 1902 is recognized as a classic.

Koltzoff early showed his independence of opinion and his slight respect for the traditional authorities either in science or in politics. He criticized the medieval system of apprenticeship which gave a young scientist little possibility for carrying on research. His little pamphlet entitled *White Slaves* (Belye Raby) was distributed in 1910 at the St. Petersburg University Zoology Department (by the late J. A. Philipchenko, another figure in genetics in Russia) as a warning to the enthusiastic students not to expect much from connection with the faculty. Koltzoff had some trouble with the authorities following the publication of this pamphlet in spite of the fact that it was unsigned.

Crisis Metabolism

The following episode which occurred in the life of Koltzoff was characteristic of him. In 1918 or 1919 at the culminating point of the Denekin intervention, a force of cavalry under General Mamontov was approaching the city of Orel less than three hundred miles from Moscow. It was pressing close on the heels of the Red Army then in full retreat. In Moscow, opposition circles created an underground committee which chose popular leaders and persons known to all Moscow as the members of the prospective government should Moscow be taken by the White Army. Many of those chosen by this committee were decided upon without the knowledge or consent of the men themselves. Unfortunately, Koltzoff was among the popular men thus drafted for an emergency which never materialized. Consequently Koltzoff was arrested and put in prison. Here he was well treated but after a short trial he was sentenced to be shot. The sentence was not to be executed immediately and several weeks were at the disposal of the doomed scientist. In spite of the depression resulting from the prison regime, he kept his mind active not in the direction of self-preservation but in the direction of study and observation. Koltzoff obtained the permission of the prison warden to use scales and to weigh himself and all the food he consumed during the period before the trial, during the trial, and after the passage of the death sentence. He kept a record of the food and the general metabolic relations and computed the intake in terms of calories. He found that the curve of weight was first horizontal and that during the trial it went down in spite of the fact that an undiminished amount of food and calories were consumed. It went sharply down after the death sentence was pronounced and continued steadily down to the point at which he was pardoned and regained his freedom. The day he was released, Koltzoff had a heavy knapsack containing food and belongings and walked several miles during that day and the next. All the while he somehow managed to keep track of his weight and of the calories consumed. He found that in spite of his strenuous physical exercise and without any increase in the amount of food consumed, his weight remained the same at first and then underwent a considerable gain.

In 1922 the author received in this country directly from Koltzoff the inter-

esting scientific document resulting from the above observations. It had been published in Moscow on a very poor grade of yellow paper and bore the impersonal scientific title "The Balance of Human Weight During An Unstable Equilibrium." This document which had no political implications whatever is a splendid example of the reaction of a thoroughly intellectual scientist, one who did not cease to work under any circumstances but who continued to observe, to experiment and to interpret.

Genetics and Eugenics

Another difficulty which Koltzoff had during the present regime was connected with his daring statement as to the origin of a certain famous Russian physicist of the Eighteenth Century. I refer to Lomonosov who was born in Kholmogory near Archangel. Doubting his proletarian origin, Koltzoff pointed out that Kholmogory was a place of exile of the nobles, the Boyars, who had displeased the Muscovite Tsars in the past. This remark made in connection with a discussion of eugenics was extremely unpopular in the official circles of Moscow. So also was Koltzoff's open advocacy of eugenics which later he had to give up. Nevertheless, Koltzoff was against reactionary and racist types of eugenics. This was well illustrated by the enthusiasm with which he displayed to geneticists the pedigree of the great poet Pushkin who counted among his eight great grandparents people of almost as many different nations, including Abyssinia.

Koltzoff has been the central figure in the rise of an important group of workers in theoretical genetics in the U.S.S.R., including originally A. S. Serebrovsky, V. S. Kirpichnikov, N. P. Dubinin, N. Timofeef-Ressovsky, Heinrich Friesen, S. L. Frolova, B. L. Astaurov, G. Gershenson, I. I. Romashov, V. V. Sacharov, B. N. Sidoroff, A. N. Promptov, G. G. Tiniakov and many others. He had for a time, in the genetic education of these investigators, the help of B. S. Tshetverikoff. Associated with the group more recently, in addition to some of the above names, have been I. E. Trofimov, V. V. Khvostova, I. N. Sveshnikova, N. N. Sokolov, A. A. Gavrilova, G. Papalaschwili, E. N. Volotov and E. I. Baskatina. In the period of greatest material difficulties occurring during the civil war, Koltzoff had gathered many of the first mentioned scientists around him and inspired them to a resumption of their scientific work. He encouraged them especially to prosecute work with Drosophila, because of the comparative simplicity and inexpensiveness of the equipment and food material required for it, combined with the high theoretical interest of the work that could be done. He realized from the first the correctness of the interpretations that had already been given by the American school of Drosophila workers. Koltzoff continued throughout his life to be an active influence in stimulating the younger generation and even in his later years he made it a point to visit each worker in his institute each day to discuss the individual working problems. The true spirit of scientific cooperation and keen interest in the programs of the work pervaded his institute.

According to information received from U.S.S.R. in the past two years, the authorities decided about two years ago to discontinue genetic research in Koltzoff's institute, and to have the remainder of the institute, reorganized under the title of "Institute of Morphology and Physiology of the Cell," taken over by the Academy of Sciences.

Salivary Gland Chromosomes

In 1934, Koltzoff, very soon after Painter's discovery was announced, originated — independently of Bridges and several months before him — the theory of salivary gland chromosomes as polytene structures which has since been proved to be essentially correct.* "The first step," I quote T. S. Painter, "towards a clearer understanding of the

*"The Structure of the Chromosomes in the Salivary Gland of Drosophila," *Science*, 1934, v. 80.

nature of salivary chromosomes was made by Koltzoff and Bridges, independently, when they attempted to account for the great size of these elements in the fruit fly. They suggested that the giant chromosomes represented uncoiled prophase chromosomes which have undergone a number of longitudinal divisions without an accompanying cleavage of the nucleus of the cytosome. In effect each salivary chromosome is a bundle of chromosomes."

In the year 1923 the first issue of the "Journal de Biologie Experimentale," edited by Koltzoff, was published. This was carried on under the same name until 1931. In 1932 the name was changed to the "Journal de Biologie" (Biologicheskii Zhurnal). Koltzoff was joined by B. P. Tokin as co-editor and the editorial staff was enlarged to include such authorities as J. J. Schmalhausen, H. J. Muller, M. M. Zawadowsky, A. S. Serebrovsky and A. A. Zavarzin. This journal has now ceased to exist, the last issue received in this country being that of 1938, Vol. 7, No. 5. In this issue one may read the following editor's note: "With the first number of 1939 the "Biologicheskii Zhurnal" will be called "Zhurnal Eksperimentalnoj Biologii" (Journal of Experimental Biology). It will be one of the Biological Series of the publications of the Academy of Sciences of the U.S.S.R."

Last Papers

More recently, Koltzoff redirected the attention of general biologists and geneticists to the problem of the lampbrush chromosomes in Amphibian oögenesis and published extensive studies and interpretatitons of them, in terms of the activities of their contained genes. In this he was following up views which he had expressed earlier concerning the nature and the functions of the chromosomes.

The last two papers of Koltzoff which were published in the "Comptes Rendues (Doklady) de l' Academie de Sciences de l' U.RS.S." (1940, 28, No. 6) deal with melanophores, a problem he had studied for many years.

Koltzoff's scientific interests covered many fields and he was not a narrow specialist. His main interest, however, was in the analysis of the physiology of heredity, in the structure of protoplasm and in the nature of living matter.

The wife of Dr. Koltzoff, M. P. Sadovnikova, was his devoted comrade and helper. She was especially interested in the inheritance of temperamental traits, and conducted experiments on this subject was rats, using objective tests. Three reports of her work were published by her in this country, in the *Journal of Comparative Psychology* and in the *Journal of Experimental Zoology*, in 1923 and 1926 respectively. After most of this account of Dr. Koltzoff was written, the undersigned received information that she too had died. According to this very recent information, in the middle of November, Professor and Mrs. Koltzoff went for a month's stay to Leningrad. Professor Koltzoff worked in the National Library on a report "Morphology and Chemistry" which he was going to deliver February second on the 145th Anniversary of the oldest Moscow scientific society, the Society of Naturalists. He fell ill suddenly of heart disease and after four days of intense suffering he died on December 2. Mrs. Koltzoff "could not survive him," and died 15 hours later. The funeral took place in Moscow on December 6.

D. N. Borodin

New York

Utopia or Something

(*Continued from page 346*)

the very economic Hell to pay. That keeps everybody pretty busy writing checks, and everything is hunky-dory.

There is a deal of fascinating historical material, and some of the Inwin's analyses are challenging, no doubt about it. But in the end the reviewer believes the above is a fair statement of Unwin's thesis. Maybe it would work. Since accumulation of goods has been an important social factor in the past, it might not!

On the biological side, it also seems that things have been illusionarily complicated. There are two kinds of society, polygamous and (theoretically) monogamous. Polygamous societies are so busy having a good time that they never get around to burn with evangelical enthusiasm. They leave their neighbors alone and just rusticate and let tomorrow take care of itself. Monogamous societies are different. Monogamy generates stresses, and these are translated into social energy—a sort of sociological heat-engine business. It all gets so complicated that everybody starts thinking about their neighbor's iniquities and shortcomings until they end by carrying a new evangel to the ends of the earth. Anything to get away from home (buttressed with at least one highfaluting motive) is the way Unwin apparently envisions the situation. He considers that this terrific urge is to be going places and doing things to one's neighbors incident to monogamy is of the utmost social importance and all one has to do to attain a permanent march toward assumedly bigger and better things is to establish a state of perpetual (or as perpetual as possible) dual blessedness and we all will be on the beam toward Utopia. When one considers the examples Unwin cites of kind of things that monogamous societies do to their neighbors, one wonders whether progress at such a price could eevr be justified in the long run. The curious situation in Germany at the moment, where a nation appears to be dominated by a group of mad homosexuals,* to guess by the Rhoem affair and by Hess' painted toenails, must be taken into account too in the blueprinting of Utopia. Plenty of social energy seems to have been generated in Germany but whether it is due to the monogamous habits of the ruling gang might be very much a question. Where would Goering fit into that picture?

Like the economic solutions offered, one can't help but suspect that our author has gotten the cart before the horse a bit. And if monogamy is upsetting as he makes it out to be to world economy, probably it is just as well that this state of dual blessedness has never been successfully attained in any country for any length of time, for so our author assures us. There is an excellent and fascinating history of the development of common-law and ecclesiastical concepts of marriage which urgently needs to be widely read. There's also much other historical material of no little interest. But history is a large and uncontrolled grab-bag out of which one can pull almost any illustration one needs to point a moral.

That changing marriage customs to increase their kinetic energy will open the door to Utopia seems a hopeful assumption in the light of the explosive possibilities suggested by our author. One gets the impression that it is somewhat like traveling on a rocket ship, which may be all right for interstellar spaces. But after all, Utopia must be located somewhere on this mundane sphere if it is going to be much use to human beings. And one ends up wondering a bit whether pyrotechnical locomotion is likely to be the way that men will finally arrive at that blessed haven of which all reformers see just beyond one or two more out-of-jointednesses set right.—R. C.

*See *New England Journal of Medicine* for a most interesting account of angle of the Nazi war-machine as revealed by captured *luftwaffe* pilots.

EPONYMS OF HEREDITARY DISEASES

MORTON D. SCHWEITZER
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KEELER'S publication, in a recent issue of this JOURNAL,[10] of proper names applied to medicogenetic conditions has prompted me to add a number that I have accumulated during the past few years. While the use of eponymic designations is generally, and wisely, deplored, there has been no indication that the practice is diminishing and it therefore seemed desirable to add to Dr. Keeler's pioneer effort, in the hope that other investigators will do likewise.

The recognition of diseases from the names of their discoverers is often a matter of no little difficulty, and there are many reasons for this. Many of these diseases are rare and have been only little studied; they receive relatively little mention in the standard medical dictionaries and source books. Furthermore, new types are continually being recognized. For example, Pyle[12] described an unusual developmental disease affecting the metaphyses of the long bones. Recently, Bakwin and Krida[1] described the same disease in a brother and sister. Before long, we may look forward to the description of this disease as Pyle's disease or Pyle-Bawin-Krida disease. Eponymic designations may easily be extended to more and more traits. Taste deficiency might well be called Fox-Blakeslee-Snyder deficiency;[5,13] and the blood differences called Landsteiner, Landsteiner-Levine, and Furuhata,[6,15] etc. Another complicating factor is that the clinical unity of some diseases is in time cast into doubt and others may fall into disuse (e.g., von Jaksch's disease, Marie's cerebellar ataxia). Furthermore, "deuces are wild" as to the ordering of discoverers in coining eponyms, so that varied catenations of names may or may not be attached to the same or to different diseases (Rendu-Osler-Weber). Nationalism also adds complications, as French, English, Russian, German, and American eponyms may be coined to describe the same disease.

Among the familial diseases, where the factor of heredity is in need of study, there occur relatively large numbers of transitional and aberrant types. Still another source of confusion is that, not infrequently, different eponyms are applied to various parts of an hereditary syndrome. For example, Pringle's disease refers to adenoma sebaceum of the face, which is only one of the symptoms of epiloia (Bourneville's disease). Also, the same individual may be lending his name to more than one condition (e.g., Osler, Charcot, Marie), and it is not always the most common one which is the hereditary type. There are four different Pick's diseases, each named for a different physician. Only two of them are hereditary (see Dorland,[9] p. 432). Just to add one more complication: the term following the proper name may be pathognomonic in some cases (Addison's disease is quite different from Addison's anemia), and in others it may be of no importance (e.g., Marie's disease and Marie's syndrome may both refer to acromegaly). And of course the authorities often disagree.

Sometimes the name in current use does not apply to the case originally studied. For example, Dresbach's anemia is sickle cell anemia, but the case originally studied by Dresbach in 1904 is now regarded as ovalocytosis, a nonpathologic trait.[7]

Inasmuch as medical knowledge concerning many of the diseases here cited is often fragmentary, any listing of eponyms is bound to be unsatisfactory in certain particulars. In the following listing I have added a suggestive reference to the genetic literature, and occasionally a medical source. Where my eponymic designation does not coincide with those in the literature cited, the reference indicates the sources I have used; names that occur in the medical literature, but without genetic connotations, have not been included. It will be seen that information regarding the mechanism of

heredity is uncertain in many cases. Inasmuch as the latest edition of Baur-Fischer-Lenz (5th, 1940) is not generally available, citations are also made to the 1931 edition whenever suitable.

References to the literature cited at the end of the article are by superior figures (e.g. B.-F.-L.[2] = Baur-Fischer-Lenz, 1931, English translation). In cases where there are additional references to articles dealing specifically with the conditions described, these are included in the parenthesis at the end of each description. It is the author's experience that to avoid confusion it is of the utmost importance to include such detailed references in descriptive lists of this kind.

ADDISON'S ANEMIA — Pernicious anemia. Also known as BIERMER'S A.; EHRLICH'S A (?). Believed related to achlorhydria and hypochromic anemia. Responds promptly to liver therapy. Non-hereditary type due to gastric cancer. Achlorhydria from birth, other symptoms develop in childhood or adult life. Familial occurrence in majority of cases. Dominant? (B-F-L[3]: pp. 242-248. — Heath, C. W. The interrelation of pernicious anemia and idiopathic hypochromic anemia. *Am. J. Med. Sci.* 185(3):365-381. 1933. — Wilkinson, J. F., and W. Brockbank. The importance of familial achlorhydria in the aetiology of pernicious anemia. *Quart. J. Med.* 24 (94):219-238. 1931.)

AXENFELD-KRUKENBERG SPINDLE — A vertical spindle-shaped brownish-red opacity on the posterior surface of the cornea. More common in females. Heredity uncertain. (Waardenburg,[14] p. 160.)

BATTEN-STEINERT-CURSCHMANN'S DISEASE — Myotonia atrophica with cataract. Same as Delage's disease (see Keeler[10]). (Waardenburg,[14] p. 298. — Mayer, L. L., and J. A. Luhan. Myotonia atrophica with cataract. *Arch. of Neur.* 30:810-823. 1933.)

BEHR'S DISEASE — Optic atrophy with ataxia, mental deficiency, other symptoms. Onset congenital or early childhood. Probably recessive. (Waardenburg,[14] pp. 460-461.)

BEST'S DISEASE — Atrophic degeneration of the macula. Usually congenital. Irregular dominant. (Waardenburg,[14] p. 358.)

BIELSCHOWSKY-JANSKY DISEASE — Late infantile form of amaurotic family idiocy. Onset during 3rd or 4th year. Duration about four years with fatal termination. Recessive? (Waardenburg,[14] p. 376. — Hassin, G. B., and A. H. Parmelee. Amaurotic family idiocy (Tay-Sachs type). *Am. J. Dis. Child.* 35(1):87-102. 1928.)

BIERMER'S ANEMIA — See Addison's A.

BLOCH-SULZBERGER'S DISEASE — Incontinentia pigmenti: pigmented macules of characteristic size, color, form, and distribution. Occur principally on trunk. Sometimes with nail, skin, or other anomalies. Limited to females. Congenital and familial. Hereditary? (Sulzberger, Marion B. Incontinentia pigmenti (Bloch-Sulzberger). *Arch. of Derm. and Syph.* 38:57-69. 1938.)

BOURNEVILLE'S DISEASE — Epiloia: sebaceous adenoma with epilepsy and mental defect. Other effects. Expression variable. Onset childhood. Dominant with high mutation rate. See also Pringle's disease (Keeler[10]). (Gunter, M., and L. S. Penrose. The genetics of epiloia. *J. Genetics* 31:413-430. 1935.)

BROCQ'S PSEUDOPELADE — Alopecia cicatrisata: various sized and shaped patches of alopecia with atrophy of the scalp in the areas. Onset in adult life. Several familial reports. Hereditary? (Cockayne,[8] pp. 355-356.)

CAGOTS — Hypotrichosis et dystrophica unguium. Nails thickened or absent; hair fine and scanty, slow growing. Other skin effects. Hair changes may not be noted until puberty. Dominant. (Cockayne,[8] p. 238. — Clouston, H. R. A hereditary ectodermal dystrophy. *Canad. M. A. J.* 21(1):18-31. 1929.)

CATLIN MARK — Bilateral fenestrae in the parietal bones (Goldsmith). Cockayne believes identical with aplasia cutis congenita, although some cases do not have the typical hairless patch of skin over the fontanels. Irregular dominant? (Cockayne[8], p. 314— Pepper, O. H. P. and E. P. Pendergrass. Hereditary occurrence of enlarged parietal foramina. *Am. J. Roent.* 35:1-8, 1936 — Goldsmith, W. M., Bilateral Fenestrae in the Parietal Bones. *J. Hered.* 32:301-309. 1941.)

CELSIUS' VITILIGO. Also known as JONSTON'S ALOPECIA. — Alopecia areata: baldness occurring in sharply defined patches, leaving the scalp smooth and white. Requires toxic or psychic factor for its expression. Onset in childhood or early adult life. Dominant? (Cockayne, pp. 353-355. — Goodman, M. H. Relation between pigmentation and growth of hair. *Arch. of Derm. and Syph.* 40(1):76-81. 1939.)

CHAUFFARD — See Minkowski-Chauffard.

COPPOCK CATARACT—See Doyne's discoid cataract.

CROUZON'S DISEASE — A form of cranio-facial dysostosis with pronounced oxycephaly, optic atrophy, enlargement of the sella turcica, and hypercalcemia. Irregular dominant? (Waardenburg,[14] pp. 69-70. — Gross, Mlle. F. Un cas de dysostose cranio-faciale héréditaire type Crouzon. *J. de Neurol. et Psychiat.* 29(8):480-485. 1929. Benedek, Ladislaus. Über die Crouzonsche Krankheit. *Dtsch. Ztschr. f. Nervenhlk.* 146(1 and 2):54-62. 1938.)

CURSCHMANN — See Batten-Steinert-Curschmann.

DEJERINE — See Landouzy-Dejerine.

DIMMER — See Haab-Dimmer.

DOYNE'S CHOROIDITIS — Honeycomb-like large white patches in the choroid (and retina) extending to the papilla. Onset during adult

life. Heredity uncertain. (Waardenburg,[14] p. 357.)

DOYNE'S DISCOID CATARACT. Also known as COPPOCK'S CATARACT—Circular-shaped cataract localized in the posterior (or central) layers. Familial cases. Irregular dominant? (Waardenburg,[14] p. 281.)

DRESBACH'S ANEMIA. Also known as HERRICK'S A. — Sickle cell anemia: characteristic anemia with splenic changes occurring in (a small minority of) individuals whose erythrocytes exhibit in vitro sickling. Etiology of the anemia is unknown. The sickling trait is present from birth and is hereditary — probably simple dominant. (Pollack, L. H., and W. Dameshek. Elongation of the red blood cells in a Jewish family. *Am. J. Med. Sci.* 188(6):822-834. 1934.)

DUCHENNE-LANDOUZY TYPE — See Landouzy-Dejerine.

DUPUYTREN'S CONTRACTION — A contracted condition of the fingers and palm. Occurs mainly among men. Onset in middle age. Often occupational in origin. Possibly hereditary in some cases. (Schröder, C. H. Die Vererbung der Dupuytrenschen Fingerkontraktur. *Arch. f. Rassen-u. Ges.-Biol.* 28: 353-380. 1935.)

EHRLICH'S ANEMIA — See Addison's A.

FLAJANI'S DISEASE. Also known as PARRY'S DISEASE — Same as Basedow-Grave's disease (see Keeler[10]).

FRIEDREICH'S ATAXIA — Failure of muscular coordination with characteristic gait, speech impairment, other effects. Spastic ataxia may be a variant of Friedreich's ataxia, both occurring in the same family. Onset in childhood. Majority of pedigrees recessive, some probably dominant, a few sex-linked recessive. Partial sex-linkage may be present in a few of the recessive pedigrees. (Haldane, J. B. S. Partial sex-linkage of recessive spastic paraplegia. *J. Genetics* 41:141-147. 1941.)

FUCHS' HETEROCHROMIA — Heterochromia with opacities of lens and vitreous body. Other eye defects. Onset childhood or later. Heredity uncertain. (Waardenburg,[14] pp. 209, 214.)

GROENOUW'S DISEASE — Bilateral nodular opacities of cornea. Onset at puberty. Dominant. (Waardenburg,[14] pp. 152, 157. — Groenouw, A. Knotchenformige Hornhauttrübung vererbt durch vier Generationen. *Klin. Mon. f. Augenhlk.* 90:577-580. 1933.)

HAAB-DIMMER OPACITY — Lattice shaped opacity of cornea. Usual onset puberty, may also occur later. Dominant? (Waardenburg,[14] p. 153.)

HAPSBURG LIP — Progenia: lower-jaw prognathism. Heredity uncertain. (Keeler, Clyde E. Heredity in dentistry. *Dental Cosmos.* Dec: 1147-1163. 1935.)

HARLEQUIN FETUS — Ichthyosis fetalis. A fetus prematurely born with severe ichthyosis. Fatal. Believed recessive. (Cockayne,[8] p. 161.)

HEINE-MEDIN DISEASE — Infantile paralysis. It is caused by a filtrable virus, but the mode of transmission of the organism has not yet been definitely determined. Called also acute anterior poliomyelitis. Occurs mainly in children. Some evidence for hereditary susceptibility, but mechanism of heredity not known. (Zinsser, Hans [Ed.]. Virus and Rickettsial Diseases (Symposium). Cambridge, Mass.: Harvard University Press, p. 570. 1940. — Aycock, W. L. Autarceology of poliomyelitis. *West Va. Med. J.* 30(11):481-489. 1934.)

HERRICK'S ANEMIA — See Dresbach's A.

VAN DE HOEVE'S SYNDROME — See Lobstein's disease.

HUNT'S SYNDROME. Also known as PARKINSON'S DISEASE — Juvenile paralysis agitans. Occurs as sequal of lethargic encephalitis. Juvenile cases are small minority of total familial cases. Incomplete dominant. (B-F-L[3]: p. 315. — Allan, William. Inheritance of the shaking palsy. *Arch. Int. Med.* 60: 424-436. 1937. [See also Keeler[10].)

HUTCHINSON — See Tay-Hutchinson.

VON JAKSCH'S DISEASE — This name less commonly used than formerly. May refer to several types of infantile anemia, including nutritional anemia, Cooley's disease (see Keeler[10]), anemia due to congenital syphilis, etc. (Koch, L. A., and B. Shapiro. Erythroblastic anemia. *Am. J. Dis. Child.* 44(2): 318-335. 1932.)

JANSKY — See Bielschowsky-Jansky.

JONSTON'S ALOPECIA — See Celsius' Vitiligo.

KNAPP'S STREAKS — Dark brown anastomizing striae in the retina near the disk. Onset during adult life. Familial. Heredity unknown. (Waardenburg,[14] p. 426. — Batten, R. D. Angioid streaks and their relation to a form of central choroidal disease. *Brit. J. Ophthalm.* 15(5):279-289. 1931.)

KRUKENBERG — See Axenfeld-Krukenberg.

KUF'S DISEASE — Presenile type of amaurotic family idiocy with milder course. Does not result in blindness. Heredity unknown. (Waardenburg,[14] p. 376.)

LAGLEYZE DISEASE — Angiomatosis of the retina. Same as von Hippel-Lindau disease (see Keeler[10]).

LANDOUZY-DEJERINE TYPE. Also known as DUCHENNE-LANDOUZY TYPE — Progressive muscular atrophy of infants, beginning in the muscles of the face, shoulder and arm. In some pedigrees more common among men. Dominant? (Dawidenkow, S. Über die scapulo-peroneal Amyotrophie. *Ztschr. f. d. ges. Neur. u. Psych.* 122(34):628-650. 1929. — Dawidenkow, S., and N. Kryschowa. Untersuchungen angeblich gesunder Verwandter von Muskeldystrophikern. *Ztschr. f. d. ges. Neur. u. Psych.* 125(1):31-45. 1930.)

LITTLE'S DISEASE — Several clinical entities included. Most common is infantile spastic diplegia. Many due to birth injuries. Possibly hereditary in some cases. (B-F-L[3]: p. 370; B-F-L[2]: p. 411.)

LOBSTEIN'S DISEASE — Complex syndrome: blue scleras, brittle bones, dental anomalies and otosclerosis are more commonly observed. Pedigrees and individuals vary in the variety of symptoms they present. Congenital. Dominant. Also known as van de Hoeve's syndrome and Eddowe's syndrome (see Keeler[10]). (Hills, R. G., and S. McLanchan. Brittle bones and blue scleras in five generations. *Arch. Int. Med.* 59:41-55. 1937. — Ottley, C. M. Osteopsathyrosis (Lobstein's disease). *Arch. Dis. Child.* 7(39):137-148. 1932.)

LORAIN'S DISEASE — Ateleiosis. "Lilliputians." Arrested physical development of unknown causation. Several clinical types. Normal size at birth. Mechanism of heredity uncertain. (Cockayne,[8] p. 387. — B-F-L[3]: pp. 106, 132. — Hogben, L. Genetic analysis of family traits, II. Double gene substitution. *J. Genetics* 25:211-240. 1931.)

MAL DE MELEDA. Also known as MLJET'S DISEASE — Thickening of skin of hands and feet (dorsal and ventral surfaces), elbows and knees, with hyperidrosis. Onset infancy or childhood. Recent evidence supports recessive interpretation. (Bosnjakovic, S. Vererbungsverhältnisse bei der sogenannter Krankheit von Mljet (Mal de Meleda). *Acta Dermato-venereol.* 19(1):88-121. 1938. — Niles, Henry D., and Margaret M. Klumpp. Mal de Meleda. *Arch. of Derm and Syph.* 39:409-421. 1939.)

MARFAN'S SYNDROME — Arachnodactyly (spider fingers). Fingers (and toes) extremely long and thin. Occurs with dislocation of lens; often also bone, joint and other effects. Congenital. Incomplete dominant in some pedigrees?; recessive in a few. (B-F-L[3]: p. 110. — Waardenburg,[14] p. 316. — Valentin, B. Arachnodactyly (pp. 455-464) in Die Morphologie der Missbildungen des Menschen und der Tiere. Jena: Fischer. 1937.)

MARIE'S ATAXIA — Hereditary cerebellar ataxia. This includes several clinical types. As commonly used, questionably distinct from Friedreich's ataxia (q.v.). Age of onset somewhat older, more often with ocular symptoms. (B-F-L[3]: p. 361; B-F-L[2]: p. 415. — Waardenburg,[14] pp. 472-473.)

MARIE'S DISEASE (A) — Acromegaly. Enlargement of the bones and soft parts of the hands, feet and face, associated with overgrowth (especially tumors) of the pituitary body. Onset during adult life. Sometimes familial. Recessive? (B-F-L[3]: p. 276; B-F-L[2]: p. 320. — Curschmann, H., and J. Schipke. Über familiäre Akromegalie und akromegaloide Konstitution. *Endokrinol.* 14 (2):88-100. 1934.)

MARIE'S DISEASE (B) — Peroneal atrophy. Same as Charcot-Marie-Tooth disease (see Keeler[10]).

MECKEL'S DIVERTICULUM — Congenital pouch or cecal appendage of the ileum; a remnant of the vitelline duct. Hereditary? (Blacker,[4] p. 305.)

MEDIN — See Heine-Medin.

MEIGE'S DISEASE — Same as Milroy's disease (see Keeler[10]).

MIBELLI'S DISEASE — Porokeratosis; a skin disease characterized by hypertrophy of the stratum corneum, followed by progressive atrophy, usually circular or oval at the start. More common in males. Dominant. (Cockayne,[8] p. 153.)

MINKOWSKY-CHAUFFARD SYNDROME — Familial acholuric jaundice. Hemolytic jaundice with splenomegaly. Probably always congenital. Spherocytosis and erythrocyte fragility are incompletely dominant. (Cheney, W. F., and G. Cheney. Chronic hereditary hemolytic jaundice. *Am. J. Med. Sci.* 187(2):191-213. 1934.)

MLJET'S DISEASE — See Mal de Meleda.

MORQUIO'S DISEASE — A form of generalized bone dystrophy marked by shortness of the body, short neck, sternum bent forward, kyphotic spine, and hips flexed in a crouching position. Onset in childhood. Hereditary? (B-F-L[3]: p. 134. — Ellman, P. A rare primary osseous dystrophy (case resembling Morquio's disease). *Brit. J. Child. Dis.* 30 (355-357):188-193. 1933.)

NIEMANN-PICK DISEASE — Severe defect of lipoid metabolism with anemia, leukocytosis, and enlarged spleen and liver. The tissues throughout the body become filled with phosphatids, which give them a foamy (microscopic) appearance. The disease runs a rapid course, ending in death within the first two years of life. More common in females. Familial. Hereditary? (Pick, L. Niemann-Pick's disease and other forms of so-called xanthomatosis. *Am. J. Med. Sci.* 185(5): 601-616. 1933.)

NONNE'S DISEASE—See Meige's disease.

OSLER — See Rendu-Osler-Weber.

PARRY'S DISEASE — See Flajani's disease.

PICK — See Niemann-Pick (See also Keeler[10]).

RENDU-OSLER-WEBER DISEASE — Hereditary hemorrhagic telangiectasis: tendency to bleeding due to local lesions of the capillaries of mucous membrane, particularly in the nose. Telangiectatic spots may be present on skin and elsewhere. Congenital. Dominant. (Pardo-Costello, V., and E. Pastor Fariñas. Hereditary multiple telangiectasia. *Arch. of Derm. and Syph.* 39:1025-1034. 1939.)

ST. VITUS DANCE — See Sydenham's Chorea.

SCHILDER'S DISEASE — Encephalitis periaxialis diffusa: a massive inflammation of the white matter of the cerebral hemispheres, beginning in the occipital lobes and characterized by early disappearance of the myelin. Occurs mostly in children and young subjects, and begins with occipital blindness. Often fatal. Heredity uncertain. (Symonds, C. P. Encephalitis periaxialis diffusa — Schilder's encephalitis. *Brit. J. Child. Dis.* 25(292/294):83-95. 1928.)

SPIEGLER'S TUMOR — Benign cystic epitheli-

oma. Usually on face or scalp. Commonly occurs at puberty or early adult life. Somewhat more frequent among females. Dominant. (Cockayne,[8] p. 301. — Wise, Fred. Multiple superficial benign epithelioma of the skin. *Arch. of Derm. and Syph.* 19:1-21. 1929.)

SPIELMEYER — See Stock-Spielmeyer-Vogt.

STARGARDT'S DISEASE — Degeneration of the macula lutea occurring before puberty. Usually recessive; one case (Halbertsma) sex-linked recessive. (Waardenburg,[14] p. 358.)

STEINERT — See Batten-Steinert-Curschmann.

STOCK-SPIELMEYER-VOGT DISEASE — Amaurotic family idiocy, juvenile type. Cerebromacular degeneration with pigmentary deposit in retina, but without cherry-red spot characteristic of infantile (Tay-Sachs) type. Onset about 6 years, slowly progressive, fatal within 10 years. Recessive. (Sjögren, T. Die juvenile amaurotische Idiotie. *Hereditas* 14:197-425. 1931. — Dide, M., and L. v. Bogaert. Sur l'idiotie amaurotique juvenile (Type Spielmeyer-Vogt). *Revue Neurol.* 69:1-42. 1938.)

STRÜMPELL — See Westphal-Strümpell.

SULZBERGER — See Bloch-Sulzberger.

SYDENHAM'S CHOREA. Also known as ST. VITUS DANCE — A symptom of rheumatic fever, whose other major manifestations are polyarthritis and rheumatic heart disease. Onset in childhood. Specific etiology unknown. Hereditary susceptibility probably recessive. (Wilson, May G. Rheumatic Fever, Ch. 3. New York: The Commonwealth Fund. 1940.)

TAENZER'S DISEASE — Ulerythema ophryogenes: a disease marked by erythema of the skin around the hair follicles, which become filled with a plug of hardened epithelium, leading to baldness. Present from birth or early childhood. Hereditary? (Cockayne,[8] p. 141. — Marx, W. Beitrag zur Histologie des Ulerythema ophryogenes. *Arch. f. Derm. u. Syph.* 163:6-17. 1931.)

TAY-HUTCHINSON'S CHOROIDITIS — Degeneration of the macula with secondary pigment deposition. Periphery unaffected. Usual onset during 5th decade. Dominant? (Waardenburg,[14] p. 364.)

UNNA'S NEVUS — A flat, nonelevated capillary nevus varying in color from pink to dark bluish-red, located chiefly in the face and neck. Heredity uncertain. (Cockayne,[8] p. 300.)

USHER'S SYNDROME — Retinitis pigmentosa with deafness. Progressive contraction of visual field (with night blindness). Deafness from birth, onset of retinitis in childhood. Individuals may show only retinitis or deafness. Recessive. (Waardenburg,[14] p. 400. — B-F-L[3]: pp. 42, 64; B-F-L[2]: p. 249.)

VOGT — See Stock-Spielmeyer-Vogt.

WALDENSTROM'S DISEASE — Osteochondritis deformans juvenilis. Same as Legg-Calvé-Perthes disease (see Keeler[10]).

WEBER — See Rendu-Osler-Weber.

WESTPHAL-STRÜMPELL'S PSEUDOSCLEROSIS — Believed related to Wilson's disease (see Keeler[10]). (B-F-L[3]: p. 365; B-F-L[2]: p. 413. — Kehrer, F. Zur Ätiologie und Nosologie der Pseudosklerose Westphal-Wilson. *Ztschr. f. d. ges. Neurol. u. Psych.* 129(3/4): 488-542. 1930.)

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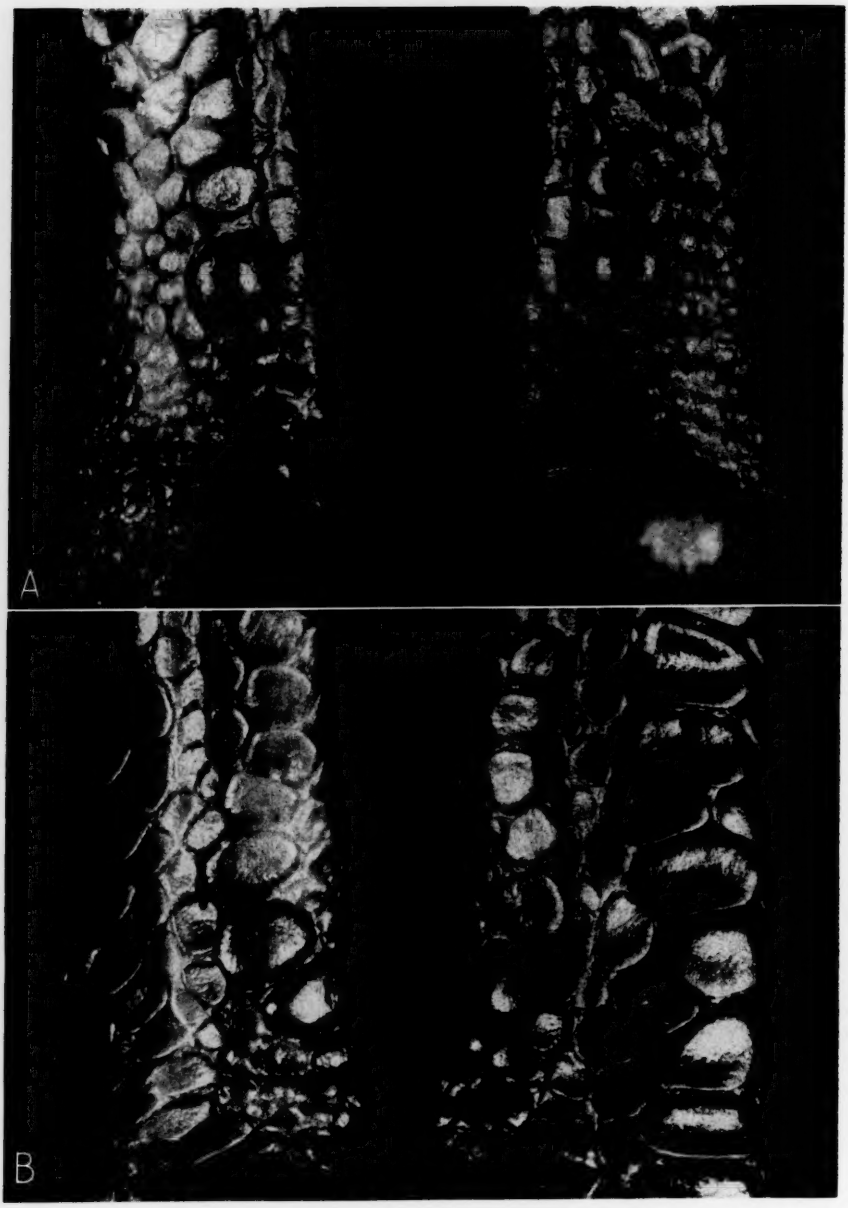

SPUR PRIMORDIA

Figure 6

At the time of hatching the spurs appear on the shanks of a newly hatched chick of a single-spurred breed as enlarged scales (*A*). A chick carrying the gene for multiple spurs shows several enlarged scales on the shanks (*B*). In the females these spur primordia will remain rudimentary (see Figure 10).

GENETICS OF THE FOWL

15. Multiple Spurs, a Mutation Linked with Duplex Comb

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IN the four decades that have passed since Bateson told the Royal Society of London how rose comb, pea comb, and dominant white are inherited, nearly all of the structural variations differentiating breeds of fowls have been submitted to thorough genetic analysis. An exception is found in the multiple spurs of Black Sumatras, which have apparently escaped the attention of geneticists because that breed is uncommon. The writer is indebted to his colleague, Dr. G. O. Hall, for pointing out that triple spurs are characteristic of Black Sumatras and for securing the stock used in the studies reported below.

Multiple-spurred cocks were mentioned by Bement[2] over ninety years ago as occurring in New York State among descendants of a Dominique cock. This antedated the introduction from Sumatra to the United States in 1847 of the fowls generally considered to be the ancestors of the present-day Black Sumatras of America and Europe (Brown[3]). Tegetmeier[10] referred to the same peculiarity in certain game fowls said to have originated in India, some of which had five spurs on each leg.

So far as the writer is aware, the mutation has not been incorporated as a breed characteristic except in the Black Sumatras. This breed resembles game fowls in conformation and is sometimes called the Black Sumatra Game (Figure 7). Females weigh four to five pounds. The plumage is greenish black. Otherwise the breed is characterized by a pea comb, very small wattles, and an unusually long tail. The multiple spurs are the only single peculiarity not found in any other breed of America or Europe. Concerning these, Brown[3] merely says that the cocks are often double-spurred, but the standards set up for the breed by the American Poultry Association[9] call for "triple spurs on cocks."

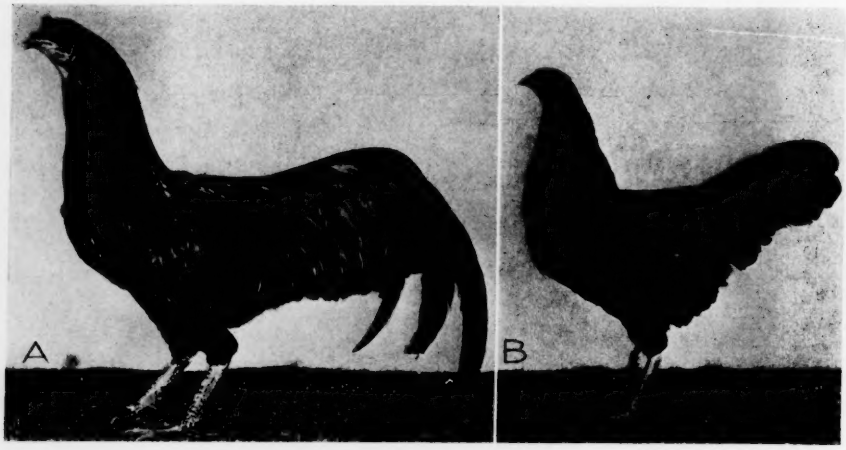
Description

Adults. Mature males have from three to five spurs on each shank. In most cases, the longest of these is the second one down from the top (Figure 8*B*) and spurs below it are successively smaller. Sometimes the spurs on one shank are almost equal in length (Figure 8*C*). Scales on the shanks below the lowest spur are usually somewhat thickened. Homozygous males seem more likely to have four or five spurs than heterozygotes.

Removal of the integument reveals a large mass of bone, sometimes of loose texture, attached to the tarso-metatarsus at the site of the spurs. The normal single spur contains a conical core of bone (Figure 9*C*). The masses underlying multiple spurs show a separate core for each of the larger spurs, but the lower part runs downward in an irregular projection (Figure 9*A-B*) that is not attached directly to the tarso-metatarsus. In extreme cases, this flange-like protuberance may go even lower than the first phalanx of the inner (first) toe (Figure 9*B*).

The adult female of single-spurred breeds shows at the site of the spur an enlarged oval scale which is framed, as it were, by smaller ones (Figure 10*A*). In multiple-spurred females, three or more scales at the spur site and below it are enlarged and usually somewhat flattened. The "frame" is not conspicuous (Figure 10*B*). There is very little chance of not recognizing the mutation in females, even though the spurs do not ordinarily develop in that sex.

Immature males. Development of multiple spurs in young cockerels apparent-



BLACK SUMATRA FOWLS
Figure 7
This breed was introduced into the United States in 1847. The males (*A*) have multiple spurs, and the females (*B*) are without spurs.

ly proceeds as with single spurs, except that there are centers of ossification in from three to five scales rather than in a single papilla. As a result, immature multiple-spurred males have a small ridge of growing spurs (Figure 8*A*) instead of the single bony knob found in their less distinguished relatives. As with single ones, the multiple spurs do not become attached to the tarso-metatarsus until there has been quite an extensive growth from the ossification centers located in the epidermis.

Chicks. This mutation is quite easily recognized in newly-hatched chicks by the presence of several enlarged, flattened scales beginning at the spur site and extending downward and toward the back of the shank (Figure 6*B*). The general appearance is quite different from that in ordinary chicks, where the single spur papilla stands out like a little round ball partially embedded in the surrounding scales (Figure 6*A*). It is not possible to recognize at hatching any consistent difference between males and females with respect to the manifestation of multiple spurs.

Classification

In two populations, each having at least half its members multiple-spurred and the remainder normal, the classifications at hatching time were verified either at maturity or at ten weeks of age when surplus males were marketed. The accuracy of the chick classifications was as follows:

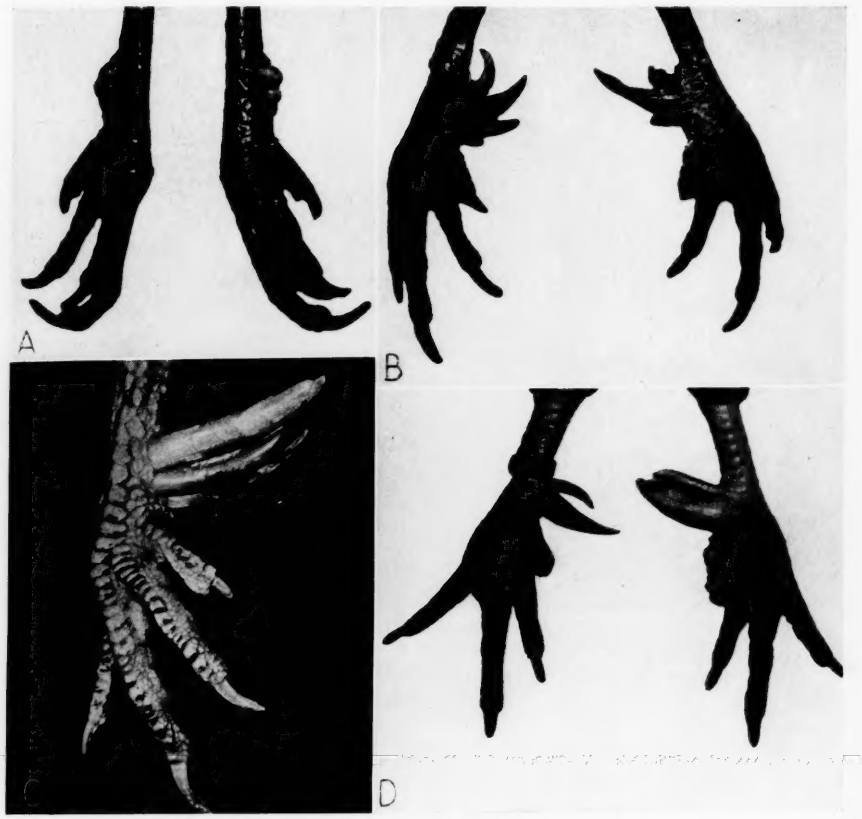
Year	Birds No.	Accurately classified at hatching per cent
1936	294	93.5
1939	266	98.9

Because of the clerical errors inevitable when one observer calls descriptions and another records them, as was done in these cases, it would be unusual for any character, even for such a sharply-defined one as rose comb, to be classified at hatching with any greater accuracy than that attained with multiple spurs in the 1939 population. Accordingly, most of the classifications in the genetic studies reported below were made at hatching.

Genetics

A. Genetic Basis

In 1935, a Black Sumatra male with typical multiple spurs was crossed with Single Comb White Leghorn females. All of the 51 birds in the F_1 generation had multiple spurs. In the F_2 generation raised in 1936, the segregation did not



BLACK SUMATRA SPURS AND OTHER TYPES
Figure 8

A—The budding multiple spurs of a young Black Sumatra cockerel, and the feet of a mature Black Sumatra male (*B*), showing four spurs on the left shank. *C*—An unusual type of multiple spurs in the Black Sumatra in which all three spurs are of equal length. *D*—These double spurs of a Barred Rock male differ in appearance as well as genetically from the other multiple spur types shown in this figure.

deviate significantly from the expected ratio of 3 multiple : 1 normal.

	Multiple	Single
Observed	79	37
Expected	87	29

$\chi^2 = 2.94$ $P = .09$

During the six-year period from 1936 to 1941, inclusive, several backcrosses were made of heterozygous males and females to single-spurred birds. In each of these there was a reasonably good fit of the observed numbers to the 1 : 1 ratio expected if multiple spurs were a unifactorial dominant character. The results were:

Year and mating	Progeny: Multiple	Progeny: Single
1936 ♂ B6005	105	102
1938 ♂ D5970	226	230
1939 ♀ E6070	3	5
1939 ♂ E5869	139	160
1940 Six ♀♀	22	29
1940 ♂ G5026	5	5
1941 ♂ H5031	41	45
1941 ♀ H4248	11	12
1941 ♂ H5022	43	55
1941 Ten ♀♀ (to date)	155	149
Observed totals	750	792
Expected (1 : 1)	771	771

$\chi^2 = 1.144$ $P = .2—.3$

It is clear from the F_1, F_2, and back-

SPUR CORES

Figure 9

The spur cores of a young Sumatra cockerel (*A*) and of an old Sumatra male (*B*) are compared with the conical core of the single spur (*C*) normal in the white leghorn male. *D*—shows the spur core of the double spurred Barred Rock whose shanks are shown in Figure 8*D*.

cross populations that multiple spurs are caused by a single dominant gene. It is proposed to designate it by the symbol, *M*, which, so far as the writer can determine, has not previously been preempted for any other mutation in the fowl. The fact that this gene is autosomal has been proven by matings of genetically multiple-spurred females with single-spurred males, which yielded female progeny showing the mutation.

B. Suppression of M

In six of the eight backcross matings

Table I. **Differential manifestation of M in the progeny of two mm males, each mated to the same four Mm females.**

Female	♂ H4999 Progeny M	m	χ^2	P	♂ H5100 Progeny M	m	χ^2	P
H4255	6	16	4.545	.036*	7	13	1.800	.186
H4260	7	23	8.533	.01 *	13	7	1.800	.186
H4263	8	26	9.529	.01 *	7	7	.000	
H4266	9	16	1.960	.171	7	11	.800	.7 - .8
Combined	30	81	24.57	.01 *	34	38	3.488	.3 - .5
Expected	55	56			36	36		

*Values of *P* showing deficiencies of *M* too great to occur by chance.

listed above that yielded more than ten offspring, there were fewer progeny with multiple spurs than with single ones. However, the deficiency was not great in any one mating, and, as the values for χ^2 and P show, even the accumulated deviation from expectation for all matings combined was not statistically significant. There is little reason, therefore, to suspect that the character was not fully expressed in all these matings. Until the spring of 1941 it was considered that multiple spurs were always clearly manifested by fowls heterozygous for *M*.

However, in that year incontrovertible evidence to the contrary was obtained in a mating, the results of which are shown in Table 1 but are not included elsewhere in the data published in this paper. Four *Mm* females used in tests for linkage of *M* were mated early in the season with one Single Comb Ancona male and, later on, with another of that same breed. The progeny of the second male, H5100, showed a close fit to the expected 1 : 1 ratio of *M* to *m*, not only for all chicks combined but also in the family from each of the four females. On the other hand, ♂ H4999 caused a shortage of multiple-spurred chickens in the progenies of all four females, the discrepancy below expectation being statistically significant in three families even in comparatively small numbers. The total number of multiple-spurred offspring was 25 less than the 55 or 56 expected in this population of 111 birds. To make assurance doubly sure, the record for this male given in Table 1 includes only birds classified at hatching, at 12 days of age, and again when about 8 to 10 weeks old. Even at the latest of these ages, some of the multiple-spurred chickens, did not show the character clearly.

This double mating shows (1) that the four females were producing in equal numbers gametes bearing *M* and *m*, (2) that the expression of *M* was unaffected by any contribution from the second male, but (3) that ♂ H4999 did contribute something, presumably modifying genes, that suppressed multiple spurs in about 46 per cent of his *Mm* progeny.

C. Linkage

Because the multiple-spurred condition is dominant, sharply distinct from the "wild type" single spur, and readily recognizable at hatching, the mutation is an ideal one for studies of linkage. Accordingly, beginning in 1936, it was tested for linkage with various genes marking each of the four autosomal link-

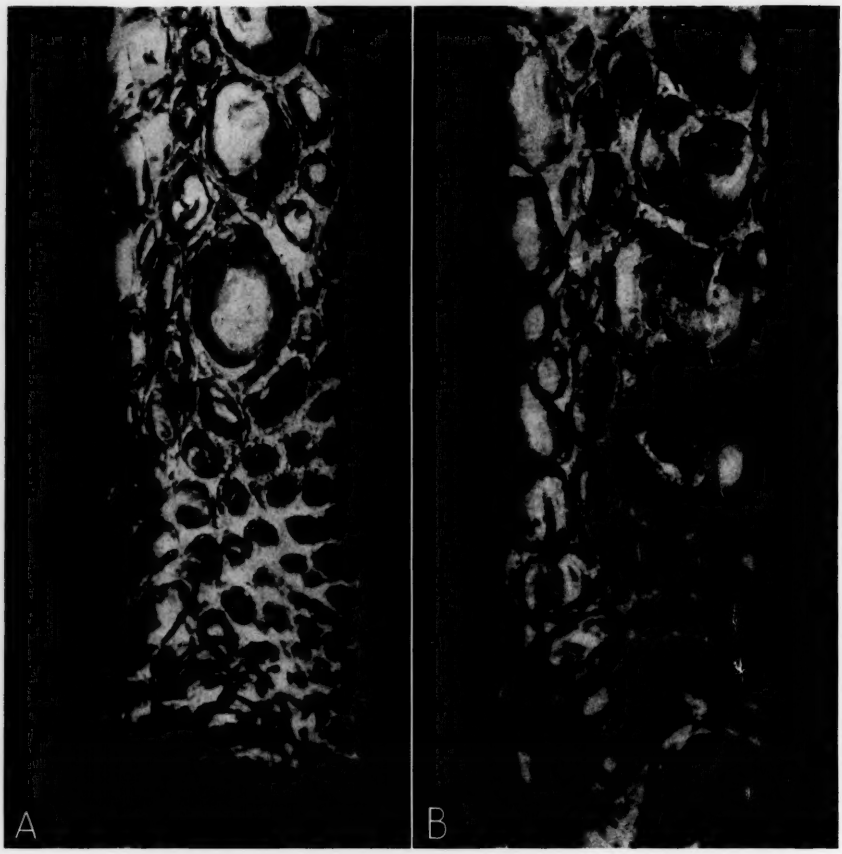
Table II. Tests for linkage of multiple spurs with known linkage groups.

Year	Group tested	Gene tested	Ratio of parental combinations to new ones*
1936	F I Cr	I	92 : 115
1941	F I Cr	I	56 : 73
1936	O P aa	P	101 : 106
1938	Op B U	B	226 : 218
1939	h F1	F1	120 : 122
1941	h F1	F1	21 : 30

* In backcrosses.

Table III. Evidence from backcrosses that multiple spurs, M, is linked with duplex comb, D.

Year	Heterozygotes tested	Classification of progeny				Gametes tested		Crossing over
		Parental classes		Cross-overs		Total	Cross-overs	
Repulsion phase		D m	d M	D M	d m	no.	no.	per cent
1939	♂ E5869	104	116	26	61	307	87	28.3
1941	Ten females	116	122	41	48	327	89	27.2
1941	♂ H5022	34	38	5	20	97	25	25.8
Coupling phase		D M	d m	D m	d M			
1940	Two females	8	20	4	9	41	13	31.7
1941	♂ H5031	34	27	18	7	86	25	29.1
						858	239	27.8



SPUR VESTIGES IN FEMALES

Figure 10

A—shows the leg of a female of a single spurred breed, with a single enlarged scale at the site of the spur. In the multiple-spurred Sumatra (*B*) there are three or four enlarged scales, giving a markedly different appearance.

age groups then known in the fowl. The results of these tests are shown in Table II. For the sake of brevity, the linkage groups are there designated merely by the symbols for the genes known in each. More complete information about them is given by Hutt and Lamoreux.[7] The writer is indebted to Dr. D. C. Warren of the Kansas Experiment Station, Manhattan, Kansas, for supplying birds carrying the mutation "flightless" used in one of these tests.

The results given in Table II show no evidence that *M* belongs in any of the four autosomal linkage groups previously known. However, clear evidence was obtained in 1939 to show that *M* is linked with the gene, *D*, causing duplex comb, or split comb. Further tests have verified this relationship and a new (fifth) autosomal linkage group is thereby established. The evidence for this is presented in Table III, which gives classifications of the progeny from a number of matings

in which birds heterozygous for *M* and *D* were backcrossed to double recessives.

In the six different matings reported in Table III, the amount of crossing over varied only from 25.8 to 31.7 per cent. Altogether, a total of 858 gametes was tested and in these crossing over was 27.8 per cent.

In the progeny from some of these matings there was a deficiency of duplex combs below the 1 : 1 ratio expected. For the three matings in the repulsion phase, the ratio of *D* : *d* in the offspring was 326 : 405, where the expectation was 365.5 in each class. The manifestation of duplex comb in heterozygotes varies all the way from a deep bifurcation to an almost invisible indentation in the posterior end of the comb. Such lesser manifestations of the gene are difficult to detect in chicks. Moreover, it is fairly certain that a number of heterozygotes are phenotypically indistinguishable from *dd* birds, and that this accounts for most of the deficiency of duplex combs in these matings. Since only 326 were observed where 365 were expected, it would appear that in about 11 per cent of the *Dd* birds the manifestation of *D* was suppressed.

In these same matings, the manifestation of *M* was practically normal, there being 348 *M* : 383 *m*, a deviation of only 17.5, or 4.8 per cent, from the 365.5 expected in each class ($\chi^2 = 1.675$; $P = .2$). Since there is no evidence in these matings that suppression of *D* was associated with suppression of *M*, there is no reason why birds carrying *D* but not showing it should be any more frequent in the parental class than in the crossover ones. Accordingly, the value of the data for determination of the amount of crossing over is not impaired by the deficiency of *D* phenotypes.

Discussion

Apart from the hereditary tendency for females to develop spurs, reported by Goodale,[5] the only mutation previously known to affect those structures in the fowl was the recessive spurlessness described by Kozelka.[8]

The multiple-spur mutation considered in this paper is not to be confused with the double spurs occasionally found in breeds other than Sumatras. Some years ago, the writer obtained from one flock of Barred Rocks two males, both of which had well-developed double spurs of the type shown in Figures 8*D* and 9*D*. Some evidence was obtained that this characteristic was hereditary, but the comparatively slow development of spurs in Barred Rocks and the fact that development of the second spur was sometimes not evident until the male was over a year old prevented an adequate genetic analysis. In these birds, as in the double-spurred Brown Leghorns described by Domm,[4] there were no more than two spurs. This condition is clearly different from the three to five spurs found as a breed characteristic in Sumatras.

Multiple spurs, so-called, are found in other species of the Phasianidae. At first glance it would be interesting to speculate on the homology of spurs in Black Sumatra fowls and in the Sumatra Bronze-tailed Pheasant, *Chalcurus chalcurus*, of which Beebe[1] says "one bird had five spurs, all others examined had two on each leg." However, from the descriptions and illustrations of this species and of others in the genera *Chalcurus*, *Polyplectron* and *Ithagenes*, all characterized by double or multiple spurs, it is evident that the extra spurs are spaced at some distance from the site of the usual single spur, and are therefore quite different genetically from the multiple spurs of Black Sumatra fowls. In one Blood Pheasant (*Ithagenes*) examined by the writer, the two spurs were over an inch apart on each shank. According to Guyer,[6] males of this genus may have as many as four pairs of spurs, and the Painted Spur Fowl, *Galloperdix lunulata*, may have two or three pairs.

No adverse effects of multiple spurs upon viability, either embryonic or postnatal, have been noted. In one mating of ten multiple-spurred females backcrossed in 1941 to a single-spurred male, infertility was only 3.1 per cent and the proportion of fertile eggs hatched was 91 per cent. The ratio of *M* to *m* in the progeny was 155 : 149. This normal

viability and the facts that the mutation is dominant, manifested in both sexes, and readily recognizable at hatching or even earlier, make the character very useful for further studies of linkage.

Summary

Multiple spurs, a breed characteristic in Black Sumatras, were found to be caused by a dominant autosomal gene, *M*. Affected males have from three to five spurs at and below the site of the usual single spur. The mutation is readily recognizable in females and in newly-hatched chicks of both sexes by the absence of the spur papilla normally occurring in single-spurred fowls and the presence of three or more enlarged and flattened scales.

Tests for linkage proved *M* to be independent of marker genes in four autosomal linkage groups, but linked with the gene, *D*, causing duplex comb. This establishes the fifth autosomal linkage group to be discovered in the fowl. The two genes show about 28 per cent of crossing over.

The mutation is useful for further linkage studies because it is dominant, manifested in both sexes, and recognizable in newly-hatched chicks with an accuracy of over 98 per cent.

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THE MICHIGAN HUMAN HEREDITY CLINIC

A DEPARTMENT of Human Heredity was organized at the University of Michigan by the Board of Regents in March, 1941. This department is a unit of the Laboratory of Vertebrate Genetics, but is closely associated with the Medical School. An Hereditary Clinic affiliated with the University Hospital will be opened in the near future. This clinic will also have close cooperation with the other departments and clinics on the campus.

The staff of the Heredity Clinic includes: Dr. C. Nash Herndon, who has previously been associated with Dr. William Allan in the Department of Medical Genetics in the Bowman Gray School of Medicine; Dr. Charles W. Cotterman, who was trained under Dr. Laurence H. Snyder at Ohio State University; Dr. Byron O. Hughes, a physical anthropologist in the University of Michigan School of Education; and Dr. Lee R. Dice, Director of the Laboratory of Vertebrate Genetics. Provision has been made for a secretary and other assistants.

Family records being secured by Dr. George R. Moore and Dr. Hughes in the Orthodentics Clinic are being deposited in the files of the Heredity Clinic. These records supply evidence of the inheritance of certain dental and jaw abnormalities. Other special human characters to be investigated by the Heredity Clinic will probably include anemias, convulsive states, deafness, racial traits, and longevity.

A large wooden frame building has been assigned by the University Hospital for the work in human heredity and this is now in the process of being equipped. Funds for the support of the investigations are being supplied by the Board of Governors of the Horace H. Rackham School of Graduate Studies.

TASTE REACTIONS OF THE DIONNE QUINTUPLETS*

NORMA FORD AND ARNOLD D. MASON
University of Toronto

THROUGH the kind cooperation of Dr. Allan Roy Dafoe a preliminary test has been made concerning the taste reactions of the Dionne quintuplets. Of prime interest is the determination of their reaction to phenyl-thio-urea, or phenyl-thio-carbamide (P. T. C.), since taste differences to this synthetic compound and related substances are known to be inherited.[3,4,5] It is well established that the population is divided into two groups, "tasters" and "non-tasters," and it has been suggested by Fox[5] that this peculiar phenomenon is due to the difference in solubility of the product in the saliva of various individuals. Among the tasters the dominant factor for taste is phenotypically variable. The majority of tasters find this substance bitter in varying degrees, while some tasters describe it as sweet, salty or sour. The explanation of these differences in taste is that the whole gene-complex of a person influences the taste-gene or in other words the genes "live in different houses." Since monozygotic multiple sets have a common inheritance and hence a common gene-complex, the members of such a set should theoretically taste alike. Their reactions to phenyl-thio-urea are, therefore, valuable indicators as to their monozygosity or otherwise.

In the great majority of uniovular sets the taste reactions are found to be similar within the set and yet instances of differences between identical twins have been recorded by Ardashnikov[1] and Rife,[4] although these conclusions are also questioned by Hartman.[7] The Dionne quintuplets who are a monozygotic set have a common heredity repeated in five persons and hence the possibility of any difference in taste between the members of the set is greatly increased.

The Tests for Taste

The Dionne quintuplets were tested for taste on April 5, 1941, when they were six years and ten months old. The tests used had previously been tried out at the St. George's School for Child Study, at Windy Ridge School, in some of the Toronto Public Schools and among students at the University of Toronto. The age levels in these tests ranged from two years to adults.

In the test each person is given a number of pieces of paper to chew, some of which have been dipped in standard solutions to give the four tastes of salty, sweet, sour and bitter. The paper used throughout the test is one known commercially as Old Vale. In dipping the paper long strips were cut, two inches in width, and these were passed slowly through the solution four times and were then hung up to dry. The strips were later cut into three-quarter inch pieces. The strengths of the solutions used were: common salt, 5.9%; citric acid, 7.8%; saccharine, 0.3%; quinine, 0.8%; and phenyl-thio-urea, 0.3%.

The only significant taste reaction in the series is that to phenyl-thio-urea, but the value of the other tests is twofold: (1) they enable one to judge how much reliance can be placed upon the answers given and (2) they assist the taster in making his comparisons. For exact measurements of taste the solutions themselves must be used, but children are naturally suspicious of a row of bottles containing fluids and hesitate to swallow a series of liquids. The dipped paper is more conveniently handled and willingly chewed by young children and hence is convenient in survey work where a simple technique is necessary.

*A contribution to the dental studies of the Dionne Quintuplets.

Procedure in Testing the Quintuplets

Each quintuplet was tested separately, with no siblings in the room. Their teacher, Mlle. Gaétna Vézina, explained to each child in French that she would be given several pieces of paper to chew and that some of the pieces had something on them to taste while others were merely paper. As each piece was handed to the child the question was asked in French, "Do you taste anything and if so, what is it?" The children cooperated excellently in the test and showed great interest and spirit.

Results of the Tests

Comments made by the quintuplets during the test are summarized below. Notes by way of explanation recorded by their teacher and by N. Ford have been added in brackets. The names are indicated by initials: C, Cecile; Y, Yvonne; E, Emilie; M, Marie; A, Annette.

1.	Paper dipped in common salt (salty)	C. Cela goute "l'hostie." Y. Non. E. Cela goute la gomme. M. Non. A. Goûte crême forte (Musterole).
2.	Plain paper	C. Non. Y. Non. E. Non. M. Non. A. Non.
3.	Paper dipped in citric acid (sour)	C. Goûte le sirop blanc pour le rhume (Pas bon). Y. Non. E. Ce n'est pas bon du tout. M. Pas bon du tout. A. Oui, c'est fort (Fait la grimace).
4.	Plain paper	C. Non. Y. Non (Semble plus certaine). E. Non. M. Ne goûte rien. A. Non.
5.	Paper dipped in saccharine (sweet)	C. Comme du sirop sucré pour rhume. Y. Non. E. Goûte les bonbons de Maman. M. Pas de goût. A. Oui, goûte le bonbon.
6.	Plain paper	C. Non. Y. Non. E. Non. M. Non. A. Non (Response expression).
7.	Paper dipped in phenyl-thio-urea	C. Ce n'est pas bon (A definite reaction). Y. N'aime pas le goût (Fait la grimace). E. N'aime pas le goût—pas bon. M. N'aime pas le goût du tout. A. Oui, c'est fort. (Grimace, very expressive.)
8.	Plain paper	C. Goûte "l'hostie" encore. Y. Non. E. Pas bon au goût. M. Non. A. Cela goûte le papier.
9.	Paper dipped in quinine (bitter)	C. Goûte le sirop d'érable. Y. Non, n'aime pas cela (Douteux). E. Pas bien bon (Grimace). M. Pas bon (Grimace). A. C'est fort.

Discussion

It has been our experience that a test for taste is a type of test with which a child is usually unfamiliar and therefore at a first trial some children are far less responsive than others, but with a second trial an unresponsive child may react very differently. Many adults find it difficult to describe their reactions to these tests or else fail to experience any taste. The latter is particularly true of the salty paper and the former of the citric acid. Children more than adults have a strong "carry-over" from the phenyl-thio-urea and thus frequently ascribe some taste to the plain paper which follows this in the series of tests (test No. 8).

Although the tests to the quintuplets were given rather hurriedly and were not repeated, we judge that their sense of taste is keen and compares favorably with that of children of their own age. For comparison with the comments made by the quintuplets, a summary is given in Table I of a random selection from our files of tests given to seven-year-old children. It will be noted that at this age level children do not use abstract terms, such as sweet or sour, but give the name of some substance known to them and also that a frequent remark is "I don't know" or merely "No," when they are unable to make a comparison in taste. Because young children are less familiar with tastes than older persons and because of their difficulties of language it has been the rule of some investigators[2] to exclude from their records children under ten years.

On this first occasion of testing, Cecile was the most imaginative, Yvonne and Marie the most reserved. Cecile compared the salt to the "host" (familiar to her in the Roman Catholic mass), citric acid to unpleasant cough medicine, saccharin to sweet medicine, and quinine to maple syrup. Each child was very definite concerning her reaction to phenyl-thio-urea.

Conclusions

From these tests it appears that the Dionne quintuplets are all "tasters" for phenyl-thio-urea. The unpleasant reaction they experienced was clearly shown in their expressive faces, while the explosive remarks which followed, such as, "Je n'aime pas la gout du tout!" left no doubt regarding the matter. Their mother also experienced a decidedly bit-

TABLE I.—Taste Reactions of Children, Seven Years of Age, Selected at Random
(Comments on tests in which plain paper was used have been omitted)

	Salt	Citric Acid	Saccharine	Phenyl-thio-urea	Quinine
Joan C.	I taste something	Lemon	Don't know	Ugh! I hate the taste	Something on it
Billy B.	Salt	Don't know	Something like sugar	Not anything	Do not like it, not very much
Frank R.	Pepper	Jelly	Jelly	Like cake, stiff cake	Like ink
Billy L.	Salt	Vinegar	Vinegar	Nothing	Salt (spit it out)
Peter W.*	Salt	Don't know	Don't know	Nothing	Don't know
Bobby W.*	Salt	Don't know	Sugar	Nothing	I have never tasted that. ugh!

*Monozygotic twins.

ter reaction. Whether or not non-tasters occur among the siblings remains to be determined.

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◆

The Beatification of the House Mouse

THE title of this comprehensive book* might well have specified the one species *M. Musculus,* for the book deals exclusively with this one species. Bare mention is made a couple of times of *M. bactrianus,* but without elaboration.

It is doubtful if a better informed or more experienced group of men and women could have been picked to compile the fund of of information contained.

The opening chapter on the embryology presents much new material in the development of the mouse connecting scattered bits of information known previously. Much of the histology is also new and presents the painstaking work of Miss Fekete; the photomicropraphs add much to the description.

The chapter on infectious diseases brings together much valuable material of use not only to the ordinary mouse breeder, but also students interested in pathology. The division of the chapter into Bacterial, Fungus and Virus disease is commendable. However more information on symptoms would be desirable.

The Staff of the Jackson Laboratory may be pardoned for devoting nearly one-third of the book to tumors. But on the other hand they are to be commended for the fund of information presented. This technical material is given in somewhat tabular paragraphs too condensed for the average lay reader, but invaluable to the student of cancer. The several chapters dealing with the influence of environmental factors, endocrines, etc., on tumors give the reader an insight into the complicated factors involved in the study of tumors.

The chapter on parasites, by Dr. Heston is a valuable addition and adds much to the usefulness of the book to mouse breeders and mouseries.

The final chapter on the care of mice gives an ideal set-up which may be useful under certain conditions of ample funds and help, but it is doubtful if such rigid sanitation is necessary or efficient for the practical breeder.

The book on the whole is well written, and fills the need for a reference book. It is to be highly recommended to not only mouse breeders but also to medical students for information on the neoplasms. The illustrations are excellent, particularly the photomicrographs.

The extensive biographies at the end of each chapter and the complete index make the book especially valuable to the student.

Wm. H. Gates
Louisiana State University

*Biology of the Laboratory Mouse—by the Staff of the Roscoe B. Jackson Memorial Laboratory—with a chapter on Infectious Diseases of Mice by J. H. Dingle, Harvard Medical School. Blakiston: Philadelphia, 1941.

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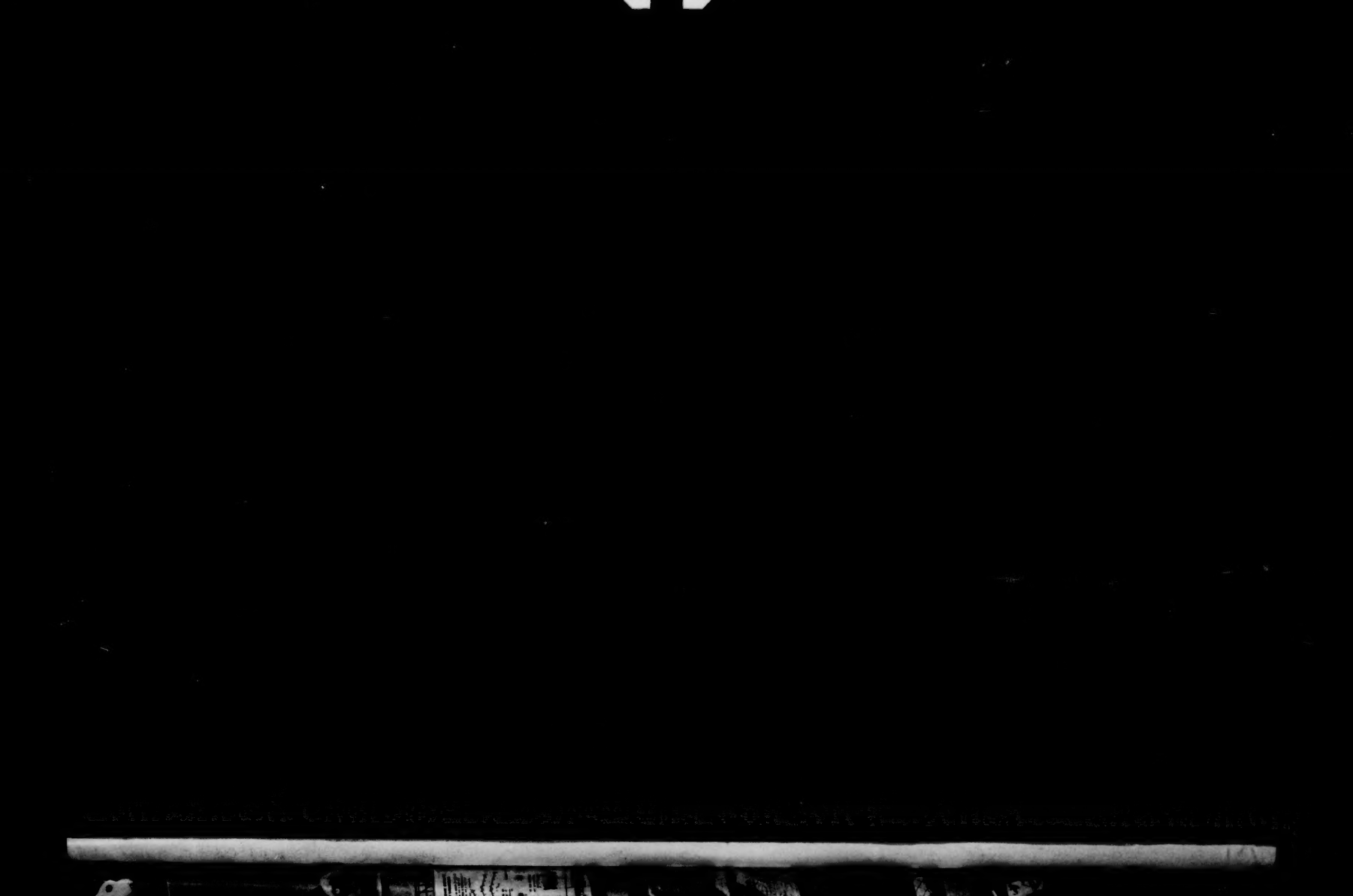
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